

Prince Edward Island

Motor neuron disease in a horse

Equine motor neuron disease (EMND) is characterized by muscle weakness, tremors, and generalized muscle atrophy (1). Differential diagnoses generally include myopathy/myositis, botulism, or equine protozoal myelitis. Diagnosis of EMND is increasing in frequency in the United States and Canada due to recognition of clinical signs and the development of ancillary diagnostic tests. This is the second reported case of EMND in the Atlantic provinces (2).

A 6-year-old, quarter horse stallion was admitted to the Atlantic Veterinary College with a history of weight loss, muscle trembling, and an abnormal stance of 2 to 3 wk duration. A complete history revealed a possible progression of clinical signs, beginning with incoordination as a yearling. No other horses on the farm were affected. No vaccination program had been instituted on the farm.

On presentation, the horse was tachycardic (80 beats/min), tachypneic (28 breaths/min), with sweating and muscle fasciculations, primarily over the neck, shoulder, and thorax. The horse was weak and most comfortable when in sternal or lateral recumbency. When he was recumbent, the muscle fasciculations were barely visible and the horse rested his chin on the ground. Neurological examination revealed no evidence of ataxia or proprioceptive deficits.

Pertinent diagnostic tests included determination of serum vitamin E concentration and peripheral nerve biopsy. **Serum vitamin E concentration was low (0.5735 $\mu\text{mol/L}$) compared to a normal horse from the same farm (2.085 $\mu\text{mol/L}$).** This is consistent with findings in similarly affected horses at Cornell University (3). Researchers there suggest that lack of antioxidant vitamin E, in conjunction with a neurotoxic insult, may lead to free radical damage of neurons, illustrating a possible etiological factor of EMND.

In this horse, biopsy of the accessory spinal nerve (ventral motor branch of cranial nerve IX), obtained under general anesthesia, reveal axonal loss and degenera-

tive changes, consistent with a peripheral neuropathy or lower motor neuron disease, such as EMND.

Based on the test results and the poor prognosis for recovery, the horse was euthanized at the owner's request. At necropsy, there were no significant gross lesions. Microscopic postmortem changes were restricted to the nervous system and paralleled those of the nerve biopsy. Varying degrees of neuronal degeneration and necrosis were noted histologically in the ventral horn of the spinal cord at the level of the cervical and lumbar intumescences. Spinal nerve roots and many peripheral nerves showed axonal and myelin sheath degeneration. There was no histological evidence of degeneration atrophy of the skeletal muscles. These findings were again consistent with a diagnosis of EMND.

Horses with EMND may stabilize with respect to their condition or may even recover; however, the prognosis for return to a functional life is grave due to residual muscle atrophy (1). Diagnostic tests for EMND, such as serum vitamin E levels and peripheral nerve biopsy, are easily performed. These tests, along with the recognition of EMND as a differential diagnosis for clinical signs of weakness, muscle tremor, and muscle atrophy, will create a great database that may aid in the discovery of the etiology and treatment of this debilitating condition.

References

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Québec

Bovine digital dermatitis

The recent examination of skin biopsies from lame dairy cattle has permitted us to suspect the presence of bovine digital dermatitis, or hairy heel warts, in cattle in Québec. It appears that the disease is widespread throughout the province, since similar lesions have been reported from the diagnostic laboratories at Saint-Hyacinthe (4 cases), Ste-Foy (3 cases), Nicolet (1 case), and l'Assomption (1 case). Macroscopically, the lesion

is typically single and localized on the caudal aspect of the hind foot, just above the heel bulbs. It takes the form of a fleshy, red, exudative, very painful dermatitis, 2 cm to 4 cm in diameter, with a very pungent characteristic smell. It may also have a verrucose or hairy appearance, simulating a fibropapilloma.

The examination of smears stained by Gram's method from fresh biopsies reveals a mixed flora

with a predominance of thin, long and undulating, gram-negative organisms, very similar to spirochetes. Histologically, the epidermis is superficially necrotic or thickened, with parakeratotic and orthokeratic hyperkeratosis. Some neutrophils can be noticed in the epidermis and in the superficial dermis. In silver stained tissue sections, numerous free spirochetal organisms are consistently found in the reactive epidermis.

Skin biopsies were cultured on various media under anaerobic conditions. Spirochetes were detected within the agar of blood agar plates after 4 d of incubation, along with colonies of other anaerobic gram-negative rods. The spirochetes grew well at 42°C and were subcultured by transferring small portions of agar onto another medium. Studies are underway to further characterize the isolates, and will subsequently be reported. Electron microscopic examination of cultures has shown that the spirochetes measure 9 µm by 0.8 µm.

According to local practitioners, the condition responds well to topical treatment with tetracycline preparations. This condition is very similar to digital dermatitis described in Europe (1) and the United States (2,3). Until the fall of 1993, such a condition had not been observed in Quebec. It is well known that spirochetes may have a tendency to opportunistically invade skin

lesions, but they may also be the primary agent in some conditions such as yaws, bejel, and pinta, which are human nonvenereal treponemal dermatitides. At the present time, it is not known if the observed spirochetes are the primary agent in this digital dermatitis or are simply opportunistic invaders.

References

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Ontario

Porcine reproductive and respiratory syndrome virus identification in proliferative and necrotizing pneumonia cases from Ontario

Between the fall of 1990 and the winter of 1991, the Veterinary Laboratory Services, Guelph, Ontario, made histological diagnoses of proliferative and necrotizing pneumonia (PNP) in nine porcine submissions (1). Since tissues had been kept frozen (-70°C), samples of lung tissues from 9 pigs representing 6 of the submissions were tested for the presence of porcine reproductive and respiratory syndrome virus (PRRSV) at the Health of Animals and Food Laboratory in Saint-Hyacinthe. A cytopathic effect was observed in porcine alveolar macrophages inoculated with homogenates of lung tissues from pigs from 3 submissions. When indirect immunofluorescence (IIF) was performed using PRRSV monoclonal antibody SDOW17, bright cytoplasmic fluorescence was observed for each isolate. Fluorescence was also noted when macrophage-propagated isolates were later tested using monoclonal antibodies VO17 and EP147, 2 monoclonal antibodies reported to react with the U.S. but not the European isolates of PRRSV (2). The reactivity of these monoclonal antibodies by IIF suggest that the PRRSV isolates from Ontario show a closer antigenic relationship to U.S. than to European isolates.

Aggregates of viral particles surrounded by gold granules were observed by immunogold electron microscopy (3) for each of the isolates from Ontario that were identified. Immunohistochemical detection of PRRSV and influenza virus type A antigens was per-

formed by immunogold and silver staining (IGSS) on formalin-fixed, paraffin-embedded, lung tissues (3,4) for 5 of the 6 submissions. Labelling for PRRSV antigens was observed in lung tissues from which the virus was isolated and also in 1 case from which PRRSV was not isolated. In addition, influenza viral antigens could be demonstrated in 1 case from which PRRSV was isolated.

The present results indicate that PRRSV can be isolated from the lungs of pigs with lesions of PNP. Isolation was successfully performed more than 2 1/2 y after the collection of the samples, underlining the stability of this virus in tissue samples kept frozen at -70°C. Furthermore, PRRSV antigens could be detected by IGSS in formalin-fixed lung tissues from which PRRSV was not isolated, demonstrating the potential usefulness of this immunohistochemical method. Although swine influenza virus could not be isolated from any of the submissions described here, influenza virus type A antigens could be demonstrated by IGSS in tissues from which PRRSV was isolated. It could not be established whether the influenza virus type A antigen represents classical H1N1 swine influenza virus or the swine influenza A virus variant reported to be the cause of PNP. The results from these Ontario cases are in agreement with those of an earlier study from Québec, in which the isolation and identification of PRRSV from lungs with lesions of PNP were reported (3). No influenza virus could be isolated from the lungs in that study, nor could influenza virus antigens be demonstrated by immunohistochemistry in lung tissues that had been fixed. In a subsequent immunohistochemical retrospective survey of PNP cases from Québec, PRRSV